

# MODULATION OF SPASTIC ANKLE STIFFNESS DYNAMICS WITH VOLUNTARY CONTRACTION IN SPINAL CORD INJURY

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## ABSTRACT

A parallel-cascade system identification technique was used to examine the intrinsic and reflex contributions to overall ankle stiffness in normal (control) and spastic spinal cord injured (SCI) subjects as a function of voluntary contraction level. Intrinsic dynamics were modeled as a linear, 2nd-order system relating intrinsic torque to joint position. Reflex dynamics were described by a linear, 3rd-order system relating half-wave rectified velocity and reflex-torque. Intrinsic stiffness was similar in magnitude in both groups and increased with voluntary contraction at similar rates. In contrast, reflex stiffness dynamics behaved differently in the two groups: (1) reflex stiffness gain was significantly greater in SCI than control subjects at all contraction levels, (2) the modulation of reflex gain with voluntary contraction was abnormal, and (3) the reflex frequency parameter was lower in SCIs and decreased with contraction level while it increased in controls. These differences were significant across a wide range of contraction levels with the gain difference being largest at low levels of contraction and the frequency difference being largest at high levels of contraction.

## INTRODUCTION

Spasticity is a motor disorder associated with lesions at different levels of the nervous system due to spinal cord or brain injury, multiple sclerosis, cerebral palsy, or stroke [1]. Common clinical symptoms in SCIs include hypertonia, autonomic hyperreflexia, flexor or adductor spasms, clonus, and weakness of voluntary contraction [2]. Nevertheless, *hypertonia*, an abnormal increase in muscle tone, is regarded as the defining feature of spasticity [3] that has both diagnostic and therapeutic significance [2]. In our previous studies, we used a parallel-cascaded system identification technique to quantify muscle tone in terms of ankle dynamic stiffness [4, 5]. Our findings demonstrated that reflex stiffness gain was significantly increased in SCIs [5]. We argued that this abnormality could be due, at least partially, to the inappropriate recruitment of larger motor units at low levels of contraction (0-10% MVC) where only small motor units are recruited in normal subjects. This argument was supported by our finding that reflex latency was shorter in SCIs than controls [6], consistent with faster conduction by larger motor axons.

Based on these findings we hypothesized that inappropriate recruitment of motoneurons could result in

abnormal modulation of reflex stiffness with voluntary contraction in spastic. We designed this study to test this hypothesis by examining dynamic stiffness and reflex function of the ankle joint as a function of voluntary activation level in SCIs and controls.

## EXPERIMENTAL PROTOCOL

Eight control subjects (4 females, 4 males) and nine SCI subjects (3 females, 6 males) with different degrees of spasticity were examined. Subjects lay supine with their foot attached to the pedal of a stiff, position controlled, electro-hydraulic actuator by a custom fitted fiber-glass boot. Joint position and torque were measured by transducers in the actuator. Electromyograms from the tibialis anterior and gastrocnemius muscles were recorded using bipolar surface electrodes.

A series of pseudorandom binary sequences with an amplitude of 0.03 rad and a switching-interval of 150 ms were used to perturb the ankle at neutral position (90°). PRBS trials were then recorded at tonic contractions ranging from 0 to 50% of the PF MVC, with the ankle at the neutral position. Trials were done at 3 Nm intervals up to -24 Nm. The different levels were examined in a different random order for each subject. Subjects had difficulty maintaining stable contractions at high torque levels. Consequently, only contractions less than 50% MVC and -24 Nm were included in the analysis. Plantarflexion is considered negative by convention.

## ANALYSIS METHODS

### Parallel-cascade Identification Technique

Intrinsic and reflex contributions to the ankle stiffness dynamics were separated using a parallel-cascade identification method [7], shown in Figure 1.

Intrinsic stiffness dynamics were estimated by determining the impulse response function ( $PTQ_{IRF}$ ) between position and torque.

Reflex dynamics were modeled as a differentiator, in series with a delay, a static nonlinear element (a half-wave rectifier) and then a dynamic linear element. Reflex stiffness dynamics were estimated by determining the impulse response function ( $VTQ_{IRF}$ ), between half-wave rectified velocity as the input and the reflex-torque as the output, using Hammerstein identification methods [7]. The non-linearity was found to be a half-wave rectifier, and thus the overall reflex gain was measured from the linear dynamic element.

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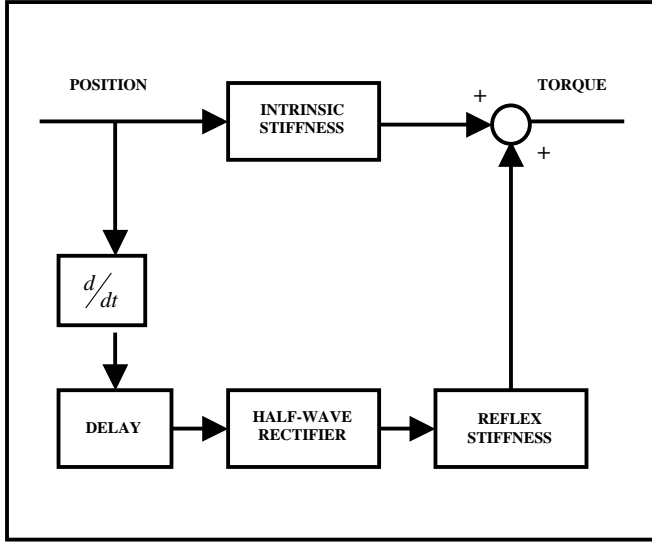


Figure 1: The parallel cascade model

### Parametric Model

Parametric estimates of the intrinsic stiffness dynamics were obtained by fitting a linear, second-order model to the compliance IRF, ( $TQP_{IRF}$ , the inverse of  $PTQ_{IRF}$ ) using non-linear least squares methods. Compliance transfer functions were considered as the second-order system:

$$TQP_{IRF}(s) = \frac{1}{Is^2 + Bs + K}$$

where  $I$ =inertia,  $B$ =viscous and  $K$ =elastic parameters.

Similar methods were used to fit a parametric model to the non-parametric  $VTQ_{IRF}$ . The linear, dynamics of the reflex stiffness were well described by a third order system:

$$VTQ_{IRF}(s) = \frac{g \omega_n^2 p}{(s^2 + 2\xi \omega_n s + \omega_n^2)(s + p)} e^{-sT}$$

where  $g$ =reflex gain,  $\xi$ =damping,  $\omega_n$ =natural frequency,  $p$  = the other frequency, and  $T$ =delay.

## RESULTS

### Intrinsic Stiffness vs Voluntary Torque (Group Results)

Figure 2 shows the group-means and standard errors of intrinsic stiffness parameters as a function of voluntary contraction. Intrinsic stiffness gain ( $K$ , Fig. 2A) and viscosity ( $B$ , Fig. 2B) were similar in both SCI and control groups; both increased as contraction levels increased. Inertial

parameter ( $I$ , Fig. 2C) was also similar in both groups and independent of voluntary contraction.

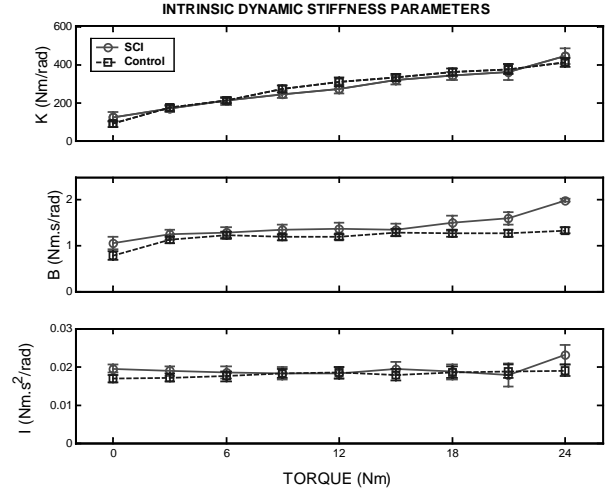


Figure 2: Intrinsic stiffness gain vs voluntary contraction. Group Mean  $\pm$ 1 Standard Error.

### Reflex Stiffness vs Voluntary Torque (Group Results)

Figure 3 shows the group-means and standard errors of reflex stiffness parameters as a function of voluntary contraction. The major findings are:

- (1) Reflex stiffness gain ( $G_R$ , Fig. 5A) was significantly larger for SCIs than for controls at all levels of contractions.
- (2) The frequency parameter ( $p$ , Fig. 5C) was smaller in SCIs than controls and the difference increased as voluntary contraction increased. Modulation of  $p$  with voluntary torque was also abnormal;  $p$  decreased as torque increased in SCIs while it increased in controls.

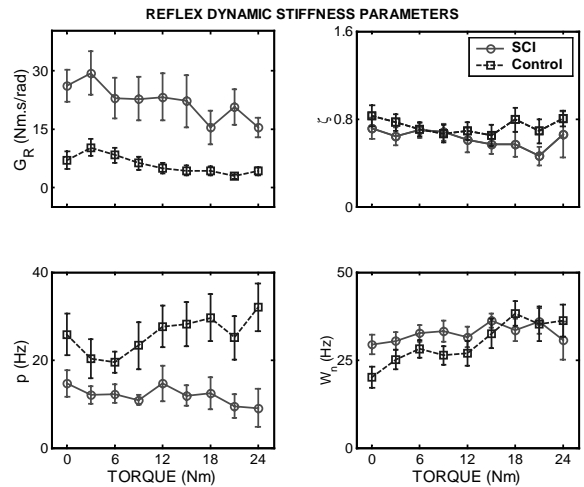


Figure 3: Reflex stiffness gain vs voluntary contraction. Group Mean  $\pm$ 1 Standard Error.

### Reflex Gain vs Voluntary Torque (Individual Results)

The modulation of most intrinsic and reflex stiffness parameters with voluntary contractions was very consistent as indicated by narrow standard error. The SCI reflex gain showed much larger variability and consequently we examined the behavior of each subject separately as shown in Figure 4. Four types of behavior were apparent

- (1)  $G_R$  remained constant at low levels of contraction and then increased for higher level of contraction (Fig 4, S2).
- (2)  $G_R$  did not change systematically with voluntary torque (Fig. 4, S1 & S3).
- (3)  $G_R$  first increased from relaxed to lowest level of contraction then decreased as torque increased, and again increased (Fig. 4, S4 & S8).
- (4)  $G_R$  changed similarly to (3) except it did not increase at the end. This trend was similar to the trend of control subjects (Fig. 4, S5-S7).

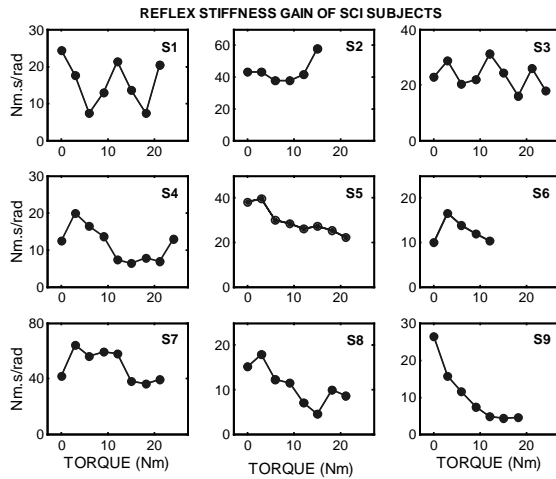


Figure 4: Reflex stiffness gain vs voluntary contraction for SCI subjects. Each panel shows the results of one SCI subject.

### DISCUSSION AND CONCLUSION

The results show that the intrinsic and reflex stiffness strongly change with voluntary contraction in both groups and demonstrate four major abnormalities in SCI subjects: (1) reflex gain was enhanced significantly; (2) modulation of reflex gain with voluntary contraction was abnormal; (3) the frequency parameter was reduced, (4) modulation of the reflex frequency parameter with torque was abnormal. In contrast to reflex stiffness, intrinsic stiffness was similar in both SCI and control groups.

The increase in reflex gain in SCI subjects may be attributed to inappropriate recruitment of motor units in spastic subjects [6], as mentioned in introduction.

Abnormal modulation of reflex stiffness gain with voluntary contraction can be caused by abnormal changes in force generation of spastic muscles due to inappropriate

recruitment of motoneurons, or perhaps a disruption in size principle.

The smaller values of the reflex frequency parameter,  $p$ , in SCIs for active condition could be attributed to neural mechanisms. Our earlier study demonstrated that, in active conditions, reflex activation dynamics (including both receptor responses and motoneuron dynamics) of both SCIs and controls are comprised of a main peak at a latency corresponding to that of a monosynaptic reflex arc, followed by a few additional bursts of decreasing amplitude at intervals of about 140 ms [8]. The amplitudes of the extra bursts were significantly larger in SCIs than controls and had inverse relation with the value of  $p$ . This suggested that repeated activation of the motoneuron pool, represented by extra peaks in the reflex activation dynamics, could be the origin of the reduced frequency parameter.

Our finding that the frequency parameter of the reflex stiffness increased with torque in controls is consistent with the size principle and the fact that higher-threshold motor units, recruited more at higher levels of contraction, have a shorter twitch contraction time. However, inappropriate recruitment of larger motoneurons and abnormal changes in neural mechanisms may cause a decrease in the frequency parameter rather than increase.

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